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AMERICAN ACADEMY OF DENTAL MEDICINE

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"To promote the study and dissemination of knowledge of the cause, prevention and control of diseases of the teeth, their supporting structures and adnexa and related subjects; and to foster and promote a better scientific understanding between the fields of dentistry and medicine."

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THE IMPORTANCE OF THE STUDY OF THE LOCAL METABOLISM IN PERIODONTAL DISEASES†

ALFONSO LENG, D.D.S., *Santiago, Chile**

It has been well established that malocclusion is one of the principal causes of periodontal disease through excessive stresses placed on the teeth. The lack of compensation of the supporting tissues is another important factor. The local metabolism of these supporting tissues may be altered when the teeth are submitted to abnormal work, and the lack of compensation may be produced. This lack of compensation may also be brought about by great systemic disturbances as well as small changes in the acid-alkaline balance, small endocrine or nutritional disturbances which are incapable of producing visible disease but act because of their chronicity at the tissues where there is some local disturbance.

All this would be but mere words if we do not prove it by experimental means. It is easy to understand these changes where inflammation exists but it is important to be aware of them where the irritational factors are not present.

At our clinic of periodontia we began to study the acid-alkaline balance in 1930. We found that the inflammatory forms of periodontal disease were inclined toward the acid side while the noninflammatory forms tended to the alkaline side.

In 1935 with Pascal Gonzalez Labbé and Julio Cabello we found that phosphatase was augmented in the bones with periodontoclasia. With Leonidas Correa, we found that when there was but little bone resorption the phosphatase was augmented in the local blood and not in the general stream; when there was a great amount of bone resorption the augmentation of phosphatase was found in the blood of the whole organism.

Because the living tissues maintain their structure by means of oxidative processes, and since the oxidation is either by virtue of the O_2 carried by the red blood corpuscles, or indirectly by the removal of hydrogen, we tried to measure the consumption of O_2 in normal and pathologic gingivae in the Warburg Respirometer (Table I). We also measured anaerobic glycolysis (Table II) and tested for the presence of lactic and succinic dehydrogenases (Table III). The dehydrogenases are important for they

Table I

RESPIRATORY ACTIVITY OF THE GINGIVAE

(*Warburg Respirometer*)

	Q_{O_2}
Normal	10.65-4.51
Hypertrophic	8.40-8.20
Inflamed	8.0 -7.60
Periodontitis	8.0 -7.0
Periodontosis	7.0 -4.0

indicate the presence of mechanisms for the transportation of hydrogen. These enzymes consist of a prosthetic group or coenzyme, which is the active portion, and of a protein carrier. The coenzymes, diphosphopyridine nucleotide and triphosphopyridine nucleotide, contain nicotinic acid, adenylic acid, and a phosphorylated pentose. The hydrogen that is picked up by the coenzyme from the substrate is transferred to flavoprotein (which contain Vit. B_2) and then to cytochromes *b*, *c*, and *a* and finally to cytochrome oxidase where it joins with molecular oxygen. The process ends with the formation of water.

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† Thanks are due Walter W. Wanio, M.S., Ph.D., and George J. Witkin, A.B., D.D.S. for their assistance in editing this paper.

Vitamin B_1 is present in a coferment, cocarboxylase, which governs the fate of CO_2. Vitamin B_2 is also important in the metabolism of protein. Thus we can easily understand the great importance of Vitamin B complex.

Table II

FERMENTATION

(*Warburg Respirometer*)

	Anaerobic $Q\frac{N_2}{CO_2}$
Normal	4
Hypertrophic	80
same treated	10
Atrophic	0.5

With Fernando Cruz and Croxatto we measured the amount of proteolytic ferments in normal and pathologic gingivae and in the granulation tissue of intrabony pockets. In normal gingivae the hypertensinase activity of proteolytic ferments is 43%, in the inflamed gingivae 54% and in the granulation tissue 73%. We believe that the organic matrix of the bone may not be indifferent to the action of these proteolytic ferments.

Total cholesterol has also been studied in our clinic and it seems to be slightly augmented in the pericementum in non-inflammatory forms of periodontal disease. It has yet to be determined if this occurs in the whole tissue or just in the blood vessels.

Table III

DEHYDROGENASE ACTIVITY

	Lactic	*Succinic*
Normal	+	+
Hypertrophic	+++	+++
Inflamed	++++	++
Control	——	——

We also measured the local amount of lactic acid in normal and pathologic gingivae (Table IV). The amount of glucose and prosphorus was locally measured by comparing the blood of the gingivae and of the finger in cases involving gingivitis, hypertrophy and paradentosis.

According to the findings of some authors, it is stated that the local disturbances of the supporting tissues can at times be of a functional nature that promotes structural changes; at other times structural changes produce functional disturbances. Concerning the last theory, if it could be proven that there were no local factors acting primarily, we might have a better knowledge of the nature of the primary structural changes of the periodontium. To establish this, there are many difficulties, for almost invariably we find local factors present that may easily explain the whole process. We believe that it is important to study those cases where the local factors do not produce any changes. These cases are scarce but there are some nevertheless.

Table IV

LACTIC ACID

(*Method of Müller-Muntz*)

Normal gum	18 mg. %
Normal blood	10-20 mg. %
Hypertrophied gum	36 mg. %
Periodontosis complic.	37 mg. %
Periodontosis atrophic	9 mg. %

It is easy to recognize the severe local difficulties and to see their effects, but the slight local or systemic disturbances are only recognized because of their constant and permanent action, as the dropping of water will, in time, wear away a stone. In this case the structural changes are anatomicopathologically more easily recognized than the functional disturbances and hence the structural changes appear to be the primary cause.

It is curious that those so called degenerative processes are active only in certain teeth and even in one part of a tooth at times. It would be necessary then to

study the nature of these degenerative processes not only anatomicopathologically but also biochemically. This must be done everywhere. It is important to use statistics in order to establish the relationship between abnormal occlusal stress and bone resorption in a great number of cases classified by ages. We have finished some work related to this. Patients of the operative clinic were used and only patients who were believed to be free from periodontal diseases. We found a large number of cases of bone resorption without pocket formation that were visibly related to malocclusion (this is important for it is an asymptomatic period of periodontitis). It is most desirable that others repeat what we have done, for unconsciously at times we may find what we wish to find.

All these preliminary biochemical works have been performed with my students and with the technical help of the teacher and assistants of the laboratory of biochemistry at the Medical School of Santiago, and in the laboratory of physiology at the Catholic University. Patients of our clinic of periodontology of the Dental School were the subjects.

We are now continuing our researches with animals, involving the changes that may be observed in the respiration of the gingivae and pericementum in relation to vitamins, hormones and other factors.

The author wishes to emphasize that all these works do not have any importance until they have been repeated and the findings corroborated.

NUTRITION AND THE DENTIST

NEAL W. CHILTON, B. Sc., D.D.S., M.P.H., *Trenton, New Jersey*

The general practitioner of dentistry is in an excellent position to be of service to his patient in the evaluation of nutritional status. Dental patients usually return to the office for periodic inspection and examination, without waiting to become dentally ill, after the dentist has taken the pains to educate them along this line. The dentist therefore has an excellent opportunity to carefully examine the mouth and oral tissues of the patient periodically, compared with the physician who, as a rule, sees the patient only when he is ill, and therefore must spend his time examining the whole body, and not performing a detailed examination of the oral tissues.

By the same token, the mouth is readily accessible for routine examination. Other body cavities, examination of which may prove of value from the nutritional point of view, are often examined only with highly technical instruments and may present personal and social obstacles on the part of the patient.

The oral cavity, in addition, is one of the most important parts of the body in the consideration of detection of the nutritional deficiency states. The mouth and its contents present a highly varied range of responses at any one time, due to the presence of the simplest type of tissue (mucous membrane) and highly specialized tissue (lingual papillae and tooth enamel). The oral tissues are constantly being subjected to trauma and irritation by mechanical, thermal and bacteriological agents and are therefore among the first to exhibit the effects of systemic disturbances. In addition, the oral cavity is part of the gastrointestinal tract and mirrors the changes affecting that system, yet it is the only part which can readily and simply be examined.

Although it is true that any qualified nutritional observer can examine the mouth, only the dentist is in a position to determine the part played by local factors in the production of oral changes suggestive of nutritional deficiency. The writer has seen nationally known nutrition consultants examine a mouth and at-

tribute the variation from the normal to nutritional deficiency alone, without taking into account the local dental factors. Any observant dentist is in a better position to evaluate the local factors than is the best-trained medical nutritionist who knows nothing of the local factors in periodontal disease.

If one steps up to the first person he meets on the street in a city like Trenton, and asks the passer-by whether he is well-nourished, the interrogator will usually receive an affirmative answer. Of course the person is well-nourished (so he says),—he has enough money to buy good food, plenty of it is available, and besides, his wife is a good cook. Yet, if we take the opinion of the members of the Committee on Diagnosis and Pathology of Nutritional Deficiencies, Food and Nutrition Board, National Research Council, we find: "All the evidence is in agreement that deficiency states are common among the people of the United States. Most of them are not the severe acute type. Rather they are less intense in degree and very much slower in their course. Predominantly, the deficiency states here are mild, moderate or severe chronic forms. Because of their slow gradual development, their presence is commonly unsuspected. In frequency and severity they increase with age and with lowered economic level. As yet optimum nutrition throughout the nation has not been achieved; on the contrary, deficiency states are present on a large scale."

The reader is then liable to point out that in his practice, most of the patients are in the upper-income brackets, and surely must be well-nourished. Let us then quote from the Food and Nutrition Board's report: "Among 225 private patients in the upper-income levels, surveyed in Philadelphia in 1940, the following percentages had diets containing less than the Board's recommended allowances for respective nutrients: riboflavin, 77; thiamin, 76; calories, 74; calcium, 46; iron, 40; protein, 37; vitamin A, 26; ascorbic acid, 13. Seven percent of the patients consumed less than the Board's prescribed amounts of all eight factors. The following percentages of the patients had less than 50 percent of the Council's recommended levels of the various constituents: riboflavin, 26; thiamin, 13; calcium, 13; calories, 5; vitamin A, 5; ascorbic acid, 4. In the surveyed group were nine physicians whose diets were as bad as those of the other patients." And so we see that patients who might readily come into our offices need the professional opinion and guidance of their dentist for the recognition of these conditions, for, by examination of the oral cavity, its contents, and the surrounding structures, deficiencies in riboflavin, nicotinic acid, ascorbic acid and possibly iron, calcium, and vitamin D, may be discovered.

If the dentist is content to perform only the mechanical phases of dental practice, and limit his observation of the dietary habits of his patients to the local effects of non-detergent diets or diets high in concentrated and refined sugars, he is only performing a partial service for his patients. If the dentist would fulfill his obligations to the utmost, consideration of the changes in the tissues of the oral cavity which may be due to nutritional inadequacies, and close cooperation with the patient's physician with regard to these changes, should be incorporated into the routine practice of his profession.

Nutritional deficiency states have formerly been considered as "all-or-none" affairs. Either a person was ill with a deficiency disease, such as beriberi, pellagra, scurvy, rickets, etc., or else he was perfectly well. We were taught that drinking orange juice for a few days would completely cure people suffering from scurvy,—they would be up and about their work as if nothing had happened. Today, nutritional authorities agree that this is not the case, that the concept of the deficiency states embodies the idea of progressive changes which may lead to definite deficiency disease or may just continue chronically, with occasional remissions or exacerbations. In order to properly understand and treat deficiency states, we must consider the mechanism of their origin.

Nutritional inadequacy occurs as a

result of two factors, which may exist separately or concomittantly:

1. Inadequate intake of nutritional factors
2. Factors which interfere with the nutrients being taken and utilized by the body (Conditioning Factors)

Six stages have been shown to exist as a result of inadequate intake:

a. Tissue Depletion—the tissues of the body gradually lose whatever substances they have stored, when the supply is either inadequate for the body needs or completely lacking.
b. Chemical Changes occur, some of which may be observed by studying the blood chemistry.
c. Physiological Changes — some of these may be determined by testing certain body functions, such as the conduction time of peripheral nerves in thiamin deficiency.
d. Sub-Gross Anatomical Changes — these may be seen by examination of the structure of the living tissues using the slit-lamp ophthalmoscope (biomicroscope).
e. Gross Anatomical Changes—these can be seen readily with the naked eye. It is at this point in the sequence that diagnosis can usually be made by the general practitioner.
f. Classic Florid Deficiency Disease—the end results, before death, which have been described in textbooks for many years (xerophthalmia, scurvy, pellagra, beriberi, rickets, etc.)

Conditioning factors may exist which may either prevent the patient from taking adequate amounts of the nutrients or may interfere with the maximum utilization of these factors by the body, thereby leading to deficiency states.

a. Impaired Intake: This may result from ill-fitting dentures, with resultant inability to masticate food, thereby causing the patient to select food which may not contain sufficient nutrients for his requirements. Many diseases, particularly those of the masticatory apparatus and gastrointestinal tract, may interfere with eating of required food elements.
b. Increased Requirement of nutritional elements, seen in pregnancy, hyperthyroidism and in fevers of long duration, may produce an inadequacy by raising the tissue requirements above the normal intake.
c. Impaired Absorption may occur in diseases of the gastrointestinal tract, such as colitis, celiac disease, achlorhydria, etc.
d. Impaired Utilization may occur after enough nutrient elements have been eaten and absorbed, when certain types of pathology, such as liver dysfunction, are present which may prevent the element(s) from being properly utilized in the body chemistry.
e. Hastened Destruction and Excretion, in such conditions as uncontrolled diabetes, prolonged diarrhea, etc., which may prevent the body from having the elements long enough to obtain the maximum benefits from them.
f. Therapeutic Procedures may interfere with the absorption and utilization of food elements. For example: mineral oil used by patients with hemorrhoids to soften the stool, dissolves out the fat-soluble vitamins, particularly vitamin A; sulfa drugs may interfere with the utilization of members of the B complex vitamins; antacids in dyspepsia may destroy thiamin and ascorbic acid in the upper gastrointestinal tract.

We see then, that deficiency states are not as "cut and dried" as was formerly thought, and that "not eating enough" or eating the "wrong" foods may not be the only cause of nutritional inadequacies. We see too, that when we believe the patient to be suffering from a nutritional deficiency, and dietary analysis shows an adequate intake, that there is a definite possibility of a conditioned malnutrition which should be further investigated by the patient's physician. An excellent op-

portunity is therefore offered for closer medicodental cooperation, which is so often talked about, and so rarely put into practice.

The question of the velocity of the deficiency states is an important one. By "velocity" is meant whether the condition is acute, subacute or chronic in duration,—conditions which all may be occurring at the same time. The acute condition results from an acute rapid deprivation of a nutrient substance from the diet. It is characterized by a rapid development and onset, and by rapid response to therapy. The chronic state takes longer to develop, takes the typical long drawn-out course with possible exacerbations and remissions and is much slower in response to therapy. Between these two stages are many intermediate ones. It must be kept in mind that there are all degrees of chronicity upon which acute episodes can be superimposed. The acute phase usually disappears rapidly on treatment, but the patient will still remain in the chronic state, with a lowered threshold for another acute episode. This chronic state must be treated for a long time before we can hope for its elimination and the patient's return to optimal health.

It is only by an intelligent understanding of the underlying mechanisms in the production of nutritional deficiency states that we can hope to treat these conditions rationally and render the best service to our patients.

225 West State Street

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RADIOGRAPHY IN PULP CANAL THERAPY

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The advent of radiography in dentistry brought a wave of "focal infection" hysteria which threatened to drive pulp canal therapy out of existence as a rational treatment for pulp involved teeth. The reason was that the radiograph disclosed infection present at the apices of a large percentage of treated teeth.

Ultimately, radiography, which first was a threat to pulp canal therapy became its savior. The dental profession came to realize that these failures in pulp treatment were due to:

(1) Treatment of teeth which should not have been treated.

(2) Failure to clean the root canals completely to their apices.

(3) Failure to fill canals completely to the apices.

To-day, pulp canal therapy, perhaps more than any other branch of dentistry, depends on radiography for its success. The early failures, prior to the use of the x-ray machine, were due to the fact that the practitioner had no means of checking the teeth for diagnosis, root canal cleansing and root canal filling.

Every root canal case should be started with a good, clear, radiograph. The shadow of the root canal on this initial film enables the dentist to select a broach of suitable gauge to extirpate the pulp. Too thin a broach will penetrate the pulp without enmeshing it. Such a broach will also pass through the apical foramen too easily. Too thick a broach will macerate

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the pulp; will not reach the apical end of the root canal; will bind in the root canal and may possibly break. The attempt should be made to use a broach which will pass to the apex of the root canal without wedging, and which is heavy enough to cope with the quantity of pulp tissue present.

It has been our practice to procure a radiograph with the broach in position ready to extirpate the pulp before the pulp is actually removed. There is no way of assuring the removal of the entire pulp unless the broach takes hold at the apical end of the pulp. If the broach takes hold at any other point, we cannot control the point at which the pulp will break off. When the broach takes hold at the apex, the entire pulp from that point downward, will come away with it.

As early as possible in root canal treatment, a measurement of the overall length of the tooth should be obtained.

(1) This provides evidence that the contents of the entire canal have been removed.

(2) It provides a guide for all instruments used in the root canal to prevent their passage through the apical foramen. Such penetration irritates the periapical tissues causing pericementitis, and

(3) It enables filling the canal to the apex.

This measurement will act as a control for root canal files when preparing the walls of the canal; for gutta percha cones or silver wires in filling the canal; for root canal pluggers in condensing root canal fillings. From the moment we have this recorded measurement of the length of the tooth, no root canal instrument, gutta percha point or silver wire should pass out of the canal into the periapical tissues.

To obtain this measurement place a suitable gutta percha cone or root canal file into the canal up to the point you believe is the apical end of the root canal. Fasten the cone or file in position and take a radiograph. If, on developing this film, you find that the cone or file has reached the desired position in the canal, it can be removed and the length recorded on the patients card. Grasp the cone or file at the incisal edge in an anterior tooth or at the height of one of the cusps in a posterior tooth and record the distance from this point to its end.

Distortions resulting from improper angulations do not markedly affect the accuracy of this measurement. We measure the device which actually reaches the apex of the tooth. The radiograph serves only as evidence of its having reached the apical foramen. However, a simple mathematical equation will give a finer degree of accuracy and remove any discrepancy. With three quantities known, we can calculate the fourth unknown quantity by formulating a proportional equation. The length of the tooth measured on the film is to the length of the file measured on the film as the actual length of the tooth is the actual length of the file used to take the measurement film.

The objective in filling the root canal is to occupy completely the entire length and width of the root canal space with a dense mass of material. The apical third of this filling must receive particular attention, inasmuch as it is the vanguard of defense against bacterial invasion. With our accurate recorded measurement of the overall length of the tooth, we can prepare a gutta percha cone or a silver wire which will fill the root canal to the apex, at the same time filling the apical third of the root canal as tightly as possible. This will reduce to a minimum the chloropercha or silver cement needed to fill in remaining spaces.

When filling the canal with gutta percha, the walls of the canal are lined with chloropercha. Then the first measured gutta percha cone is placed in position. At this point, a radiograph is made to check its position. Upon verifying its position, the filling is condensed and completed.

When filling the canal with silver wires, a measured wire is placed in the canal and a radiograph is made to check the accuracy of its position. Then, the

walls of the canal are lined with silver cement and the silver wire is placed into position in the canal.

A pertinent suggestion concerning angulation of the x-ray machine may be helpful at this point. Radiographs made with the rubber dam in position require individual decisions as to angulations used. The presence of the rubber dam clamp changes the angle between the film and the tooth. As a result, we cannot use the vertical angulations we are accustomed to when doing an intra-oral series.

One Hanson Place

THE CORRELATION OF THE ENDOCRINES AND VITAMINS IN PERIODONTAL DISEASE*

LEON SUKIN, D.D.S., *Philadelphia, Pa.*

INTRODUCTION

The oral cavity is analagous to other body cavities in that they are all governed by the same physicochemical laws and physiologic principles. They have a common source of nutrition, and are similarly protected by the same antipathologic systems. Since the mouth benefits by the general defense mechanisms of the body, it offers a dependable picture as to the state of systemic health. Many of the oral manifestations which seem to be of local disturbance are often traced to some major systemic condition. It becomes evident that the dental practitioner must consider the oral cavity in terms of its effect on the whole body and vice versa.

In a consideration of the whole body, the close relationship existing between hormones and vitamins is becoming more and more evident. Their chemical structure as well as their physiologic and clinical effects reveal to us an ever increasing conviction that life cannot exist without these substances. Even more, insufficiency or faulty utilization on the one hand, and over-abundance or excessive utilization on the other, result in definite clinical disease entities.

Hormones and vitamins alike act as catalyzers. Their chemical and functional similarities, are often remarkably close. Their physiologic activities often run parallel either in the same direction as synergists, or in opposite directions as antagonists. We must realize then, that a consideration of the vitamins is essential when dealing with endocrine problems and that both are necessary in the etiology, diagnosis, treatment and cure of abnormal conditions whether they be oral or systemic.

ORAL MANIFESTATIONS OF VITAMIN DEFICIENCIES AND THEIR DETERMINATION

VITAMIN A

Oral manifestations in its deficiency are: (1) atrophy and metaplasia of enamel and dentin, (2) keratinization of the gingivae, (3) hypertrophy of the gingivae, (4) slow recurring necrotic gingivitis when accompanied by mechanical irritations such as calculus or overhanging fillings (Sinclair), (5) poor calcification and delay in eruption of the permanent teeth, as well as thick poorly calcified jaw bones and alveolar processes (Melanby), (6) irregularity in the arrangement of the lower anterior teeth.

Laboratory Tests—Deficiency is best detected by biophotometric examinations of the eyes, also by laboratory tests employing blood serum or urine. Blue color reaction is produced by the vitamin and antimony trichloride.

VITAMIN B_1

(THIAMINE HYDROCHLORIDE)

Oral manifestations in its deficiency are (1) sensitive and painful oral tissue, (2) herpetic lesions which are often found on the buccal mucosa and palate

* Presented as a requirement for Periodontia postgraduate course, 1946, New York University College of Dentistry.

or beneath the tongue, (3) temperomandibular joint discomfort, (4) dry socket, (5) aphthae.

Laboratory Tests—A complete absence of B_1 in the urine denotes a deficiency.

No ill effects have been reported due to an excess.

Vitamin B_2 (Riboflavin)

Oral manifestations in its deficiency are (1) fissures and macerations at the angles of the mouth, (2) degenerative crustlike formation of the epithelium of the lips—most marked on the lower lip, (3) receding gingivae, (4) gingivitis, (5) glossitis in which the tongue is purplish red or magenta colored.

Niacin (Nicotinic Acid)

Oral manifestations in its deficiency are (1) scarlet tongue, (2) smooth atrophy of the lateral borders and tip of the tongue, (3) painful, sore, often swollen, fissured and corrugated tongue. It may influence redness of gingivae along with avitaminosis C. Stages from mild to acute to severe chronic may be seen.

Laboratory test—As in the case of thiamine its absence in the urine is an indication of deficiency.

An excess does not appear to produce toxic manifestations.

Vitamin C (Ascorbic Acid)

Oral manifestations in its deficiency are (1) gingivitis, (2) swollen, bleeding and livid gingivae, (3) slow healing of tissues, (4) loosening of teeth.

Vitamin C deficiency and diffuse alveolar bone atrophy have similar characteristics; that is, an inability of bone and connective tissue to withstand functional stress. The labial bone of incisor teeth breaks down first in the region anterior to the fulcrum of tooth movement. This is explained as the result of pressure on this region incidental to loosening of teeth.

Laboratory tests—Blood and urine tests are sufficiently accurate for clinical purposes and of value especially in the detection of subclinical scurvy.

An excess does not appear to produce toxic manifestations.

Vitamin D

Oral manifestations in its deficiency are (1) red marginal gingivitis, (2) alveolar bone resorption with loosening of teeth.

Other symptoms of its deficiency are (1) degenerative pulp changes, (2) improper bone formation, (3) osteomalacia, (4) osteoporosis, (5) enlargement of the joints. If the vitamin deficiency takes place during the formation of the teeth they will be poorly calcified. Where deficiency takes place during tooth eruption it may retard the eruption.

Laboratory tests—There are none available for the detection or estimation of Vitamin D in the blood or urine. Determinations of serum inorganic phosphorus, calcium and phosphatase activity, however, are helpful aids in the detection of deficiency.

Vitamin E

Its deficiency results in sterility. It affects gonadal function, causing castration-like changes in the pituitary, atrophy in the testes, and degenerative changes in the developing tissues if pregnancy does take place and is evident in miscarriage. The presence of Vitamin E seems to be essential to the activity and perhaps the synthesis of the pituitary sex hormones. Injection or feeding of Vitamin E achieves results similar to those following injection of gonadotropic hormone. The castration-like changes seen in Vitamin E deficiency, however, may be a secondary effect produced by gonadal changes.

Vitamin K

Its deficiency may result in fragility of bones, hemorrhages into joints, periosteum, intestines, muscles, etc. and lack of prothrombin in blood.

Laboratory tests—None direct, but tests are available for determining the prothombin level of the blood.

ORAL MANIFESTATIONS OF ENDOCRINE DISTURBANCES

Pituitary Gland

Hyperpituitarism is a condition due to

pathologically increased activity of the pituitary gland and marked by gigantism or acromegaly.

Oral manifestations are (1) heavy thickened tongue which in rare cases may be so enlarged as to cause the mouth to be opened permanently, (2) marked protrusion of lower jaw, (3) spacing of teeth as a result of the jaw continuing to grow after the teeth are fully developed.

Hypopituitarism is a condition due to pathologically diminished activity of the pituitary body and marked by excessive deposition of fat and the persistence or acquirement of adolescent characteristics.

Oral manifestations are irregular alignment and crowding of teeth.

Thyroid Glands

Hyperthyroidism is a condition due to excessive functional activity of the thyroid gland.

Oral manifestations are (1) alveolar absorption, (2) increased flow of saliva.

Hypothyroidism is a deficient action of the thyroid body producing myxedema or, in children, cretinism.

Oral manifestations are (1) thickening of the tissues of the lips and mouth, (2) a thick and bulky tongue which may protrude through the mouth, (3) a mucoid infiltration which may cause marked hypertrophy and puffiness of gingivae, (4) teeth may erupt in wrong sequence with improper spacing, (5) teeth may be poorly developed, (6) pyorrhea is occasionally present, (7) in the later stage there may be a tendency to develop canker sores, (8) the taste buds may develop late and function imperfectly leading often to feeding difficulties, (9) a hypersensitivity to curettage is a frequent accompanying symptom (Roth).

Adrenal Glands

In extreme hypoadrenalism due to destruction of the suprarenals, especially of the cortex, Addison's disease may result.

Oral manifestations are (1) bronzing of the oral mucosa with a continuous dark brown line near the margin of the gingivae, (2) bleeding from the gingivae, (3) atrophy of the alveolar bone may accompany this condition.

Parathyroid Glands

Hyperparathyroidism is an abnormally increased activity of the parathyroids causing loss of calcium from the bones.

Oral manifestations are (1) rarefied bone and thickened periodontal spaces, (2) giant cell cysts and tumors in the bone may develop.

Hypoparathyroidism is an insufficiency of the secretion of the parathyroid glands. Rickets may be a resulting symptom.

Oral manifestations—periodontoclasia is occasionally present.

Gonads

In hypofunction of the gonads the oral manifestations are (1) retardation of eruption of teeth and alveolar growth, (2) a congestion of the gingivae may result during menstruation, (3) alveolar decalcification and gingival hypertrophy during pregnancy.

Pancreas

Hyperinsulinism is an excessive secretion of insulin by the pancreas resulting in hyperglycemia.

Oral manifestation—there may be advanced alveolar bone destruction.

Hypoinsulinism is an insufficient secretion of insulin as in diabetes with a rise in blood sugar level.

In oral manifestations of diabetes there may be (1) marked alveolar bone loss, (2) characteristic stomatitis, (3) chronic gingivitis, (4) periodontitis marginalis, (5) paradental abscess, (6) purulent inflammation is prevailable.

FUNCTIONAL RELATIONSHIP OF THE VITAMINS AND ENDOCRINES

Vitamin A

Antagonistic tendency toward:-

1. Thyroids
2. Insulin
3. Parathyroids
4. Adrenals

Synergistic tendency with:-

5. Sex hormones and gonads
6. Gestation and lactation
7. Growth hormone

1. It has been long considered that Vitamin A is in some measure antagonistic to thyroid function. Wendt successfully treated exophthalmic goiter with concentrates of the vitamin, depressing the basal metabolism rate and improving the general condition. Iodine administration or thyroidectomy increases the vitamin level in the blood, but it is a question whether it is thyroid overactivity or exhaustion which raises or lowers the level in the blood. However, empirically, 50,000 to 80,000 units two or three times a day seem to help these patients considerably. It is noteworthy that the nervousness brought on by hyperthyroidism may possibly result in bruxism and ensuing periodontal disease.

2. In diabetes the Vitamin A content of the plasma is generally high. This is presumably due to an inability of the liver to store the vitamin since it is already over-saturated with it. In severe cases it is mainly the carotene values that are increased in the blood, suggesting that the liver is incapable of transforming it into Vitamin A. In these cases, orange-colored deposits are found in the mesenteric fat and blood vessles, indicating the possibility of a relationship between Vitamin A and diabetic arteriosclerosis.

3. Wolf states that certain features of Vitamin A activity seem to indicate their antagonism to the parathyroids, as for example its relation to the formation of renal and cystic calculi. However, clinicians who perform routine serum calcium and phosphorous examinations on patients with renal stones have uncovered several latent cases of overfunction of the parathyroids.

4. Although the modus operandi is unknown a prolonged lack of Vitamin A renders the adrenals atrophic.

5. The effect of Vitamin A deficiency upon the gonads has been studied quite carefully. In the rat the cornification characteristic of estrus is prolonged and at times even during diestrus the vaginal epithelium remains cornified. On this basis, Vitamin A therapy has been utilized in senile vaginitis. Large doses often give unusually rapid relief of the distressing symptoms concomitant with gross and histologic repair of the lining membrane. In the male the changes due to Vitamin A deficiency are even more pronounced. The terminal cells slough off at an abnormally rapid rate and the denuded seminiferous tubules gradually shrink in size. A certain amount of maturation takes place, but at a definitely reduced pace, especially in the deep layers. The maturing cells gather in nests which, when seen, indicate a Vitamin A deficiency. The effects on the gonads may be produced by way of the pituitary since it has been found that, at least in animals, a Vitamin A deficiency results in a marked increase in the basophile cells, similar to that marked after castration. Other effects of the deficiency are sterility in both men and women and most of these cases can be cured by adequate Vitamin A intake. In hypofunction of the gonads eruption of the teeth and alveolar growth seem to be retarded. Ziskin, in experiments on castrated monkeys, found that estrogenic substances such as estrin produce an increase in the thickness of the keratinized layer of the oral and gingival mucous membrane.

6. Vitamin A is essential during pregnancy and lactation. If there is marked insufficiency, intra-uterine death and resorption of the fetus due to necrosis and infection of the maternal decidua are common. Here the damage is primarily maternal, in contradistinction to Vitamin E deficiency in which it is fetal. In milder cases, gestation may be prolonged beyond term, resulting in difficult labor because of the changes in the vaginal epithelium and loss of tone of the abdominal muscles. The placenta which is often retained may show areas of hemorrhage and leukocytic infiltration. The infant mortality is high during the first few days of life on account of weakness and poor mammary function. Therefore, 8,000 to 10,000 i. u. of Vitamin A should be given to most pregnant and nursing women to insure normal physiologic function. Oral manifestations—Ziskin describes four conditions found in the gingivae of pregnant women, (1) gingival hemorrhage, (2) raspberry red gums

(small congested nodules on the gingivae, (3) generalized hypertrophy, (4) localized hypertrophy (or pregnancy tumor—Blum).

7. The influence of Vitamin A on bones and teeth is only poorly understood, but it is known that a deficiency results in an irregular functioning of the osteoblasts and odontoblasts. There is atrophy and metaplasia of the enamel and dentin, and a cessation of growth in height due to a depression in the proliferative activity of the epiphyseal cartilages. A narrow band of atrophic cartilage forms at the juction of the epiphysis with the shaft often separating the two from each other by a thin plate of bone. This interferes with the vascularization of the growing area, thus counteracting the growth impulse emanating from the anterior pituitary. These changes are almost the reverse of those seen in Vitamin D deficiency (rickets).

VITAMIN B_1 (Thiamine Hydrochloride)

Antagonistic tendency toward:-

1. Thyroids
2. Adrenals

Synergistic tendency with:-

3. Thyroids
4. Insulin
5. Prolan secretion
6. Progesterone
7. Pregnancy and lactation

1. Its antagonism is shown by distinct improvement in hyperthyroidism when Vitamin B_1 is administered. This is especially evident in the depression of the basal metabolic rate, halting loss of weight and in overcoming many of the digestive disturbances and improving the general condition.

In hyperthyroidism the classical symptoms as tachycardia, exophthalmic goiter and tremor are present. There is an increase in the basal metabolic rate which creates a nutritional need beyond ordinary requirements and an increased excretion of calcium in the urine, sometimes to as much as 250 per cent above normal. This hypercalcuria may induce an alveolar absorption because of the withdrawal of calcium salts from the bones in general. An increased flow of saliva is an important symptom.

Laboratory tests — Hyperthyroidism causes increased basal metabolic rate, decreased cholesterol values in the blood, high blood iodine values and a lowered glucose tolerance.

C. A. Mills reported that in tropical climates he noted symptoms resembling hyperthyroidism from the administration of large doses of thiamine chloride. He reports that when other parts of the B complex were added the bad effects subsided.

2. Vitamin B_1 deficiency produces hypertrophy in both medulla and cortex of the adrenals. In the cortex, numerous mitoses appear during the active period and disappear during the period of depletion. In the medulla and the sympathetic ganglia related to it, degenerative changes occur which at times are quite prominent. In this connection it might be mentioned that an adrenalin test has been employed for the diagnosis of Vitamin B_1 deficiency. Since the heart muscles are weakened, injection of adrenalin causes a typical heart attack with severe and rapid fall in diastolic pressure. However, the test is not to be used routinely since it is not without danger.

3. Synergism is indicated by the fact that both aid in the development and maintenance of the nerve structures including those of the central nervous system.

4. Both insulin and Vitamin B_1 are essential elments in the normal utilization of carbohydrates by the body. Insulin can be materially reduced when large doses of Vitamin B_1 are given simultaneously. Both are essential in aiding the disturbed carbohydrate metabolism in diabetes and in reducing the blood sugar level.

Oral manifestations in Diabetes—Polypoid proliferation arising from the undersurface of the free gingiva and usually containing pus is seen. In appearance it resembles granulation tissue and

occurs chiefly in the gingival sulcus. This is in contrast to the polypoid proliferation, but without pus, seen in non-diabetic individuals occurring most frequently at the site of the interdental papillae. Thus the location of the lesion becomes an important factor in differential diagnosis. The extensiveness of the proliferation often creates an appearance similar to that of gingival hypertrophy. In diabetes it will be noted that the free gum margin is pushed back rootward as the proliferating tissue grows and there is a general lessened resistance to pyogenic infection. Dry mouth, characteristic stomatitis, chronic gingivitis, periodontitis marginalis and paradental abscess may be present. Sometimes the tongue becomes swollen with indentations along the lateral borders. It appears thickly coated with deep painful fissures and inflamed filiform papillae. The gingivae recede and become dark red at the margin with the interdental papillae enlarged and covered with grayish ulcerations. The stomatitis may be aphthous in type or may be hemorrhagic, somewhat simulating scurvy.

5. The pituitary reacts to Vitamin B_1 deficiency by a dimunition of prolan secretion, resulting in defects in the maturation of follicles, amenorrhea and loss of libido in some cases.

6. Vitamin B_1 and progesterone run parallel in the cessation of the estrus cycle.

7. During pregnancy and lactation much more than the usual amount of Vitamin B_1 is required. When adequate amounts are taken the hemoglobin increases and there is complete freedom from annoying gastric and intestinal atony, and lactation is more satisfactory. The nervous irritability, emotional instability and signs of demineralization characteristic of Vitamin B_1 deficiency in pregnancy are effectively counteracted. However, the greatest usefulness of the vitamin during pregnancy is its power to prevent gestational polyneuritis. For supplement treatment in pregnancy, 2,000 to 4,000 Sherman units daily should be used either orally or by injection.

Vitamin C (Ascorbic Acid)

Antagonistic tendency toward:-

1. Thyroid

Synergistic tendency with:-

2. Cortin and adrenals
3. Insulin
4. Progesterone
5. Pregnancy and lactation

1. Vitamin C seems to be antagonistic to the thyroid, since it is of distinct aid in the treatment of hyperthyroidism and also because when given simultaneously with thyroxin it nullifies the activity of the latter. Thyroxin puts a definite strain upon the carbohydrate metabolism by mobilizing large amounts of glycogen from the liver and reduces the capacity of the liver and muscles to store this substance, thus causing hyperglycemia and glycosuria.

2. The adrenal cortex is an important reservoir for Vitamin C. Aside from the adrenals, rich supplies of Vitamin C are also contained in the graafian follicle, the corpora lutea, the testicles and the pituitary. Since all these glands are interrelated, this special distribution of the vitamin seems to be of considerable importance. Cortical extract is a valuable adjunct in the treatment of avitaminosis, and deficiency symptoms are delayed by its use. Cortin and Vitamin C are both linked in maintaining integrity of capillaries and both have a synergistic relation to progesterone. Both affect carbohydrate diet and both are antagonistic to thyroxin.

3. Carbohydrate metabolism is definitely affected by Vitamin C. In diabetes its administration decreases the hyperglycemia, and if given with insulin, prolongs the duration but not the intensity of its effects.

4. Vitamin C is synergistic to progesterone. The fluctuation in Vitamin C content parallels closely that of progesterone. In the corpus luteum it is found in greatest concentration in the mature state, decreasing during its involution the same as progesterone as a result of its inhibiting or depressing effect on estrin.

5. Vitamin C is also synergistic with pregnancy and lactation and scurvy rarely develops during pregnancy. The exact cause of this immunity is unknown but can be suggested by the fact that large amounts are stored in the persistent corpus lutem. During lactation, Vitamin C is needed in amounts of 80 to 100 mg. daily to pass half that amount to the infant.

Vitamin D

Vitamin D and the parathyroids are synergistic in action and both are concerned with calcium and phosphorus metabolism. They both cause a retention and concentration of these elements in the body and their deposition in the bones and teeth. Deprivation of Vitamin D will bring about compensatory hypertrophy and hyperplasia in the parathyroids. Only when the parathyroid hypertrophy can no longer compensate for a vitamin deficiency which has become too great does rickets occur. Vitamin D will produce practically all the effects of the parathormone injection.

Vitamin E

The presence of Vitamin E seems to be essential to the activity and perhaps the synthesis of the pituitary sex hormones and they are both synergistic in action. Injection or feeding of Vitamin E achieves results similar to those following injection of the gonadotropic hormone. On the other hand Vitamin E deficiency brings about changes in the pituitary resembling those seen in castration.

CONCLUSION

An attempt is made to emphasize the all important factor of diagnosis. No one can describe a universal method for complete diagnostic survey. Each patient presents an individual problem and must be analyzed separately. It is true that specific endocrine syndromes and deficient vitamin manifestations in general have a tendency to similarity, but these symptoms vary in different individuals.

Hyperthyroidism, myxedema, acromegaly and parathyroid tetany are conditions easy to diagnose if one understands the physiology of the glands which produce them. Resulting vitamin deficiencies are readily diagnosed after study and application. Again the signs produced in some patients by endocrine disorder are unmistakably characteristic.

In general, approximately 50% of the information upon which to base a diagnosis is obtainable from a thorough history, 25% results from an oral examination and the other 25% is obtainable from the laboratory tests which are confirmatory of the history and oral examination.

To obtain a relevant history one must know both normal and pathologic physiology related to the endocrines and vitamins. Especially in complete oral diagnosis the endocrines and the vitamins and their functional relationship to other organs and to each other must be known. This knowledge, together with good clinical intuition will point out the possible existent condition.

It would seem that many fields of the highest significance to the dentist are gradually opening through an increasing knowledge of the nature and functions of the endocrines and of the effects of disturbances of equilibrium among their component parts. The adverse nutritional influences on endocrine function should never be overlooked.

This closing then sums itself into the suggestion that further study may reveal still a closer correlation of the vitamins and endocrines with a more exacting and complete diagnosis for the symptoms, etiology, treatment and cure of periodontal disease.

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TOBACCO AND GINGIVITIS: The significance of tobacco in the development of ulceromembranous gingivitis and in the formation of calculus was investigated in a statistical study by J. J. Pindborg and reported in the *Journal of Dental Research* for June 1947. One thousand four hundred and thirty-three Marines between the ages of 16 and 28 were subjected to dental examinations and at the same time were questioned about their daily consumption of tobacco in the form of cigarettes, cigars, and pipe tobacco. From the results of the dental examinations, the condition of the gingivae were classified into the following three groups: (1) normal, (2) chronic simple marginal gingivitis, and (3) ulceromembranous gingivitis. Also, according to their answers on the smoking of tobacco, the Marines were divided into the following three groups: (1) no consumption of tobacco, (2) consumption of tobacco less than 10 Gm. per day, and (3) consumption of tobacco 10 Gm. or more per day. All these data were tabulated and analyzed with the following conclusions drawn:

Seemingly, the incidence of chronic simple marginal gingivitis is not affected by tobacco smoking, whereas the incidence of ulceromembranous gingivitis is markedly different in nonsmokers and smokers. Although 1.5 per cent of the nonsmokers had ulceromembranous gingivitis, 10.7 per cent of the Marines smoking more than 10 Gm. of tobacco a day had this disease. Moreover, the number of Marines with normal gingivae declines from 33 per cent in nonsmokers to 22.5 per cent in the heavy smokers.

Further, with a greater amount of tobacco smoked, there seems to be an increased possibility of developing calculus. Although 39.7 per cent of the non-smokers are without calculus, only 17.5 per cent of the persons smoking more than 10 Gm. of tobacco a day are calculus-free

On the basis of the results, nothing was inferred concerning the manner of action of the tobacco. It was assumed, however, that since tobacco-smoking may play a role in the development of ulceromembranous gingivitis, smoking should be forbidden during treatment for this condition. (Nav. Dental School, Bethesda, Md.)

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BOOK REVIEWS

Successful Dental Practice, by J. Lewis Blass, Ph.G., D.D.S., and Irwin Tulkin, D.D.S. 221 pages with 42 illustrations. Phila., Pa., J. B. Lippincott Co. 1947, $5.00.

This book is a story of how to build and maintain a successful dental practice through the intelligent practice of dentistry. It is interesting to note this new approach to practice management in which the dentist instills in the patient the desire for dental work.

The authors take the stand that most persons are interested in having a healthy mouth but are neglecting its care due to misinformation or poor relationship with the dentist. Therefore, the authors have undertaken to show how one can develop this relationship better by explanation of what work is to be done, by dispelling fears and prejudices of adults and children, by reducing emotional tensions, by treating them as human beings, by making them tooth conscious so that they want the best work possible. The thinking dentist will understand that if he resolves satisfactorily the above mentioned factors, the business end of his practice merely becomes one of simple mechanics. There are many suggestions for handling of fees, insurance, legal matters and for the general improvement of office management.

The material in the book has been gathered over many years by the authors who are well versed in the subject. Dr. Blass lectures at New York University on dental practice management and Dr. Tulkin is a lecturer in postgraduate courses on this subject. Both men have discussed this material before many dental groups.

The book is well written, simple and direct. There are numerous illustrations to help in the clarification of many problems discussed in the book. Ample material is made available to help the dentist in patient education. The book will aid in refreshing our minds with the things we know we should do, but which we have allowed to lie dormant and fall into disuse. This is a worthwhile book to read, re-read and remember. It is new, novel and a noteworthy addition to our dental literature.

L.R.B.

Signs and Symptoms Their Clinical Interpretation, by Cyril Mitchell MacBryde, A.B., M.D., F.A.C.P., 439 pages, 74 illus. Philadelphia, J. B. Lippincott Company. 1947, Cloth, $12.

How convenient it would be for the physician if the new patient were able to announce: I have a gastrointestinal disturbance," or, "My trouble is nephritis." A perusal of the usual textbooks of diagnosis or of medicine would lead one to believe this might be the case, for the chapters consider "infectious diseases," "intoxications," "deficiency diseases," "metabolic diseases," "respiratory diseases" and so on, in rigorous order, as though every sick person carried his presumptive diagnosis labeled on his chest. Where in such textbooks can the doctor seek help when the patient confronts him complaining, for example, of severe epigastric pain, or of headache, or of jaundice?

One must admit that monographic development of the complete picture of a disease is an important means of medical education, but there are serious defects in a system which encourages us to force the ailing person into a compartment, no matter how poorly it may fit him. It is widely recognized by experienced clinicians that a skillfully taken history with a careful analysis of the chief complaints and of the course of the illness, will more frequently than not indicate the probable diagnosis, even before a physical examination is made or any laboratory tests are performed. A master diagnostician I know says: "Let me take the history and I will accept any good intern's word on the physical findings." In other words, even today the accomplished physician can learn more in the majority of cases from what his patient says, and the way he says it, than from any other avenue of inquiry. If one doubts this, let him remember that pain in one of its thousands of guises is by far the most common presenting symptom. How handicapped we would be if the patient could not tell us that he had pain, or where it was, or its nature, or duration, or radiation!

A useful aid in the interpretation of symptoms consists in grouping them together to form quickly recognizable complexes or syndromes. Every medical student learns that "dermatitis, diarrhea and dementia" means pellagra and that "tremor, tumor and tachycardia" indicates hyperthyroidism. The veriest medical tyro knows that a chill, a pain in the chest and rusty sputum could hardly signify anything but pneumonia. Little medical rhymes, hallowed by word-of-mouth transmission to successive medical generations, honors ileus. "the symptom-complex known throughout the nation, characterized by pain, vomiting, tympanites and obstipation," and epilepsy, "the aura, the cry, the fall, the fits, the tonus, the clonus, the involuntary defecation." Indelibly stamped in many a physician's mind lies Charcot's Triad of insular (multiple or disseminated) sclerosis: intention tremor, nystagmus and scanning speech. Frequently, however, in the commonly recognized syndromes very little is understood as to the actual origin or mechanism of production of the primary symptoms and signs. Thus these tricks, these aids to memory, lull us into a false com-

placency, based too often upon very little real knowledge.

The physician today has many technics available to assist him in making accurate diagnoses. He can peer into the recesses of the body: bronchoscopy, gastrocopy, thoracoscopy, peritoneoscopy, cystoscopy; he can study the shadows cast by the body's part upon the x-ray film; simple x-ray, laminography, kymography, cholecystography, encephalography, gastro-enterography, pyelography, bronchography; he can remove blood or lymph or abnormal fluid accumulations for physical and chemical analysis; he can remove bits of tissue from the surfaces or the cavities of the body and study them under the microscope and in the chemical and physiologic laboratory; the action potential, the very currents of life itself, he can record and analyze in the electroencephalogram and the electrocardiogram. However, without an understanding of the meaning of symptoms, how useless are these refined diagnostic technics! They are but tools which are only as valuable as the mind which directs them. The informed mind will understand the meaning of the specific type of mucosal defect seen through the bronchoscope in relation to the patient's symptom of hemoptysis. The high icterus index observed in the laboratory study of a patient's serum has significance of one type if associated with recurrent attacks of right upper quadrant pain, but of another when found in association with a shrinking liver during pregnancy.

No mechanical measures can take the place of careful consideration of the patient's complaints. No device be it ever so clever mechanically, electrically or chemically can serve as a substitute in the art of medicine for the informed mind of the physician. The physician's ability as a diagnostician will determine the nature and the efficacy of the treatment he chooses to employ. His ability to diagnose will depend in only a minor degree upon his ability to use special technical measures. He must know when to use them, which tests to select, and how to interpret the physical findings, as well as the special laboratory tests. The physician's judgment in these matters will depend largely upon his ability to analyze and interpret symptoms.

This book attempts, so far as present knowledge permits, to give the basis for analysis and interpretation of some of the commonest symptoms which bring patients to the physician. Emphasis is placed upon the pathologic physiology of the symptom, while its correlation with other symptoms and with physical and laboratory evidence is considered as important but secondary in the diagnostic method. Our knowledge concerning many of these symptoms is incomplete, but it is rapidly expanding. Although the final word often cannot be said, critical and analytical thinking in the manner followed in these chapter should prove productive for us all: patient, practitioner, professor and student.

I. Newton Kugelmass

American Textbook of Operative Dentistry, by Arthur B. Gabel, D.D.S., M.A., et al. Eighth edition. 674 pages and illus. Philadelphia, Lea and Febiger. 1947, Cloth, $11.00.

The editor in conjunction with ten outstanding authorities in various branches of dentistry has revised the material in this edition, bringing it up to date with regard to the latest developments in the field of operative dentistry.

In addition to the purely technical phases which are excellently presented, chapters are included on Examination and Diagnosis, Caries, and Preventive Dentistry. Comparative techniques for inlay construction and evaluations of their respective advantages and disadvantages are included. The illustrations, with few exceptions, are excellent.

The reviewer finds it necessary to take exception to several statements dealing with periodontal disease which at the least are open to debate. For example the statement, "Like gingivitis, which always precedes it, periodontoclasia is essentially infective in character," does not agree with the modern concept of periodontal disease which, with the possible exception of Vincent's infection, gives to bacteria a relatively unimportant etiological role.

An illuminating discussion on the causes of halitosis (fetor ex ora) is of value to those practitioners who are so often called upon to assist in dealing with these distressing problems.

This book is of interest primarily to dental students and teachers of operative dentistry but the practicing dentist will find much that is of direct concern to him in his everyday practice.

A.N.A.

Pharmacology and Pharmacotherapeutics for Dentists, by William H. O. McGehee and Melvin W. Green. Third Edition. xx and 536 pages. Philadelphia, The Blakiston Company. 1947.

This text has been rewritten or revised in many places. Along these lines may be mentioned the material on chemotherapeutic agents, vitamins, hormones, anti-malarial drugs, and toxicology. This book may be called excellent from the standpoint that it contains within its covers information along pharmacological and therapeutic lines that is of primary interest to the dental practitioner and student. In this latter sense, the book may be regarded as complete. On the other hand, it is regrettable that more information along pharmacological lines is not presented.

Your Teeth and How to Keep Them, by Jerome J. Miller, D.D.S., with an introduction by C. Raymond Wells, D.D.S., 250 pages, 44 illustrations. N. Y., Lantern Press, Inc. 1947. $3.00.

There are many things one should know about one's mouth and teeth as individual entities or as they relate to one's general health. It is often quite difficult for a professional person to give an explanation to a layman in simple language that will be understood.

In the above mentioned book, the author has written a noteworthy text. This is a manuscript equally important to the layman and to the dentist, for Dr. Miller has brought the dental problem to the average person in terms that he can understand. The everyday questions, fears, anticipations, neuroses are explained fully and satisfactorily.

The book is well arranged and written so that it makes for easy reading. The layman can gain much useful information through the use of this book and the dentist can use it as a refresher. There are many illustrations that can be used by both these readers to good advantage. It is recommended reading for all.

L.R.B.

ABSTRACTS

Chronic Ulcerative Gingivitis and Focal Infection, by Frade Helming, L.D.S., (Copenhagen). Brit. Dent. Jour. LXXXIII; 50: Aug., 1947.

The war has brought a great increase in acute ulcerative gingivitis and altered the character of the disease, making it more violent and more difficult to cure.

There are four categories to this disease—gingivitis ulcerosa acuta, gingivitis ulcerosa subacuta gravis, gingivitis ulcerosa subacuta mites (pseudo catarrhalis), gingivitis ulcerosa chronica.

The subacute form, is often taken for catarrhal stomatitis, which, if treated at all, is treated with astringents. This has no value. Periodontal massage makes it worse. In order to differentiate between the subacute form and catarrhal stomatitis pass a blunt proble interdentally with the same pressure. There will be no pain in normal tisue or catarrhal stomatitis, but in any type of ulcerative gingivitis there is a pain reaction.

Chronic ulcerative gingivitis often resembles chronic catarrhal gingivitis but with a powerful light in the ulcerative form one can see ulcerations interdentally. Bleeding, odor, and bad taste may also be present. Pain can also be elicited on probing as described above. The catarrhal form is negative in all these tests.

Chronic forms as well as subacute and acute forms of ulcerative gingivitis may be accompanied by systemic symptoms such as depression, lassitude, etc., which must presumably be attributed to toxemia.

53 patients were examined who had chronic ulcerative gingivitis.

27 of these had systemic symptoms, primarily fatigue, but also irritation, diffuse headache, depression, etc. Many had been treated by a physician with tonics, vitamins, iron, etc., with no effect.

Of the 27 cases, 24 reacted to treatment favorably. 23 of these 24 had fever before treatment, 11 cases had a high erythrocyte sedimentation rate, all of course, had general complaints as listed above. All cases became normal after treatment with regard to general symptoms, fever and sedimentation rate except one—the sedimentation rate came down to 12 but the author points out that it was exceptionally high before treatment—28. One of the three remaining cases exhibited general complaints, fever and high ESR. He didn't respond to treatment, and was sent to a otorhinolaryngologist who found a chronic sinusitis present.

The remaining two had general complaints but no fever or high ESR, and treatment did not alleviate the complaints. Hence, the author concludes that their complaints were psychogenic.

To summarize, comparatively unpronounced chronic ulcerative gingivitis, often ignored, affects general health, and it must be attributed to bacteria, or toxins, or both, which mastication causes to pass into the blood stream.

M.M.

REPORT OF MEMBERSHIP COMMITTEE
AMERICAN ACADEMY OF DENTAL MEDICINE

DECEMBER 8, 1947

The Membership Committee met and considered the names submitted. All the following have successfully met the requirements, were recommended and elected to membership:

Guido Mercurio—Buenos Aires, Argentina
Alfonso Leng—Santiago, Chile
A. Allan Rothman—Flushing, N. Y.
R. Ronald Wyman—Boston, Mass.
John J. O'Connell—Newburyport, Mass.
Leslie Milne—Roxbury, Mass.
William F. Maguire—Boston, Mass.
Raymond E. Levisque—Gardner, Mass.
Arthur G. Lanagan—Waltham, Mass.
A. James Kershaw, Jr.—West Warwick, R. I.
Alexander L. Keltie—Westwood, Mass.
George E. Hall—Watertown, Mass.
Stanley R. Garrard—Boston, Mass.
Paul S. Finkelstein—Boston, Mass.
Charles H. Endee—Milton, Mass.
Robert Ira Diamond—Boston, Mass.
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Philip J. Burkat—Boston, Mass.
Warren L. Adams—Melrose, Mass.
Philip Stein—Bergenfield, N. J.
George Stein—New York, N. Y.
Harry Albert—New York, N. Y.
Max Shapiro—Los Angeles, Cal.
Irwin Walter Scopp—Plainfield, N. J.
William A. Rosenfield—Darien, Conn.
Salvatore T. Rifici—Jersey City, N. J.
Gunter Schmidt—St. Louis, Mo.
Dennis J. O'Donnell—Washington, D. C.
Herbert Paul Newman—North Bergen, N. J.
Samuel Mintz—Plainfield, N. J.
Robert James Miller—Louisville, Ky.
Irving E. Medoff—Newark, N. J.
Israel Harrison—Philadelphia, Pa.
William Goldring—Brooklyn, N. Y.
David Biel—Cleveland, Ohio
S. Joseph Bregstein—Brooklyn, N. Y.
Morris J. Boyer—Union City, N. J.
Samuel A. Ament—Paterson, N. J.
Edmund H. Zabriskie—Old Greenwich, Conn.
Gordon Richmond Winter—Philadelphia, Pa.
Leonard K. Vogel—New York, N. Y.
Alcide Thibaudeau—Montreal, Canada
Rowe Smith—Texarkana, Ark.
Frederick E. Sloan—Binghamton, N. Y.
John F. Deveney—Kansas City, Mo.
Martin T. Siegal—Poughkeepsie, N. Y.
Hyman Pines—New York, N. Y.
Manuel L. Matus—North Conway, N. H.
William Mulford—Newark, N. J.
Robert Felipe Mendez—Cienfuegos, Cuba
Renos M. Siman—Nicosia, Cyprus
Joseph B. Lenzner—New York, N. Y.
Carsten W. Johnson—Pleasantville, N. Y.
William Howard Hyde—Brooklyn, N. Y.
Hunter Leo Harang—New Orleans, La.
Martin Matthew Grunberg—New York, N. Y.
Joseph Ginsberg—Springfield, Mass.
Rueben Feltman—Passaic, N. J.
Herman Epstein—New York, N. Y.
Louis S. Eidinger—Montreal, Canada
Hyman Dubrow—Far Rockaway, N. Y.
Focion Febres Cordero—Caracas, Venezuela
Albert W. Canter—Jersey City, N. J.
I. Norton Brotman—Baltimore, Md.
Archie A. Albert—Pawtucket, R. I.
Harold E. Brennan—New York, N. Y.
Louis B. Biales—Cleveland, Ohio
Abraham I. Bernhak—New York, N. Y.
Martin Henry Berman—Floral Park, N. Y.
Louis Bruno Amyot—Schenectady, N. Y.
Franklin J. Goldman—South Norwalk, Conn.
Guillermo Londono—Medellin, Colombia
Nathaniel Peter Solomon—Montreal, Canada
Richard J. Behan (Assoc.), Pittsburgh, Pa.
Maurice J. Oringer—New York, N. Y.

New Jersey Section Report

The New Jersey Section of the American Academy of Dental Medicine was formally organized on October 15, 1947 at the Newark Athletic Club. An excellent dinner was held prior to the meeting which was called to order at 8:30 P.M. A motion was made and seconded to elect the Officers whose official terms would begin on January 1, 1948 and run for a full calendar year.

The following men were elected: Dr. Harold Gelhaar of Upper Montclair, President; Dr. Jack Englander of Jersey City, President elect and Dr. Lester B. Older of Union City, Secretary and Treasurer.

Dr. Samuel Charles Miller, past president of the A.A.D.M. was present and gave excellent counsel to the members.

The following men are to be considered the official Organizers of the New Jersey Section of The American Academy of Dental Medicine:

Henry Belasco
725 White Horse Pike
Audubon, New Jersey

Benjamin Lavine
751 Stuyvesant Avenue
Trenton 8, New Jersey

Abraham Allen London
714 Main Street
Boonton, New Jersey

Jacob A. Englander
2616 Boulevard
Jersey City, New Jersey

Lester B. Older
2115 New York Avenue
Union City, New Jersey

Leslie James Fitzsimmons
144 Harrison Street
East Orange, New Jersey

Samuel Harold Levine
1125 South Clinton Avenue
Trenton 10, New Jersey

Samuel Turkenkopf
195 Montclair Avenue
Newark 4, New Jersey

Harold R. Gelhaar
286 Park Street
Upper Montclair, New Jersey

ANNOUNCEMENTS

Tufts Offers Courses in Periodontology

Tufts College Dental School announces the following postgraduate and graduate courses in Periodontology:

Refresher Course I—Each Monday for the twelve weeks beginning January 19th to April 12th.

Refresher Course II—Two weeks, full-time beginning June 7th through June 18th.

Postgraduate Course III—Sixteen weeks, full-time leading to a certificate.

Graduate Course IV—Thirty-two weeks, full-time leading to the master of science degree for qualified students.

For further information write Dr. J. T. O'Rourke, Director, Division of Graduate & Postgraduate Studies, Tufts College Dental School, 416 Huntington Avenue, Boston 15, Massachusetts.

Three Periodontia Postgraduate Courses Offered by New York University

1. One week, all day—lectures and demonstrations. Fee—$150.00
2. Three weeks, all day—one week of lectures and demonstrations, two weeks of practical work on patients (class limited to 15). Fee—$300.00
3. One academic year (9 months) of lectures, demonstrations, and clinical work, leading to a certificate, (class limited to 4) Fee—$1,000.00
4. In addition, short lecture and demonstration courses will be held throughout the school year.

Apply to:

Director, Postgraduate Division,
New York University, College of Dentistry,
209 East 23rd Street
New York City, N. Y.

Post Graduate Courses Open at Ohio State

A limited number of enrollments still are available for veterans and civilian dentists in Ohio State University's post-graduate courses in dentistry being offered during March, according to Dr. Hamilton B. G. Robinson, director of the Post-Graduate Division of the OSU College of Dentistry.

Courses still open for enrollments are: Children's Dentistry, March 1-5; Endodontics, March 8-12, and Dental Caries Control, March 8-12. All courses are given for five consecutive days and are limited to 10 students. The charge for each course is $50. The courses come under the G.I. Bill of Rights. Applications should be filed as early as possible through the Post-Graduate Division of the College of Dentistry, Ohio State University, Columbus 10, Ohio.

Veterans enrolled must present their "Certificates of Eligibility and Entitlement" from the Veterans Administration when they register. Civilian dentists are required to send in a deposit of $25 with their applications.

Annual Meeting of American Academy of Oral Pathology

The American Academy of Oral Pathology will hold its Second Annual Meeting at the Hotel Stevens, in Chicago, on Sunday, February 8. Among the speakers will be:

Dr. Carl Waldron, University of Minnesota
Dr. B. O. A. Thomas, Washington University, Seattle
Dr. Edward Stafne, of the Mayo Clinic
Cdr. Carl A. Schlack, Naval Medical Research Institute
Dr. Hamilton B. G. Robinson, Ohio State University
Dr. Barnet Levy, Washington University, St. Louis
Dr. Irving Glickman, Tufts College Dental School
Dr. Henry Goldman, Boston, Massachusetts
Dr. Lester R. Cahn, Columbia University
Dr. Herman Becks, University of California
Dr. Myron Aisenberg, University of Maryland

The Academy will cooperate with the New England Society of Oral Surgeons and the Seminar of Oral Medicine in sponsoring a new periodical starting January, 1948. The new Journal of Oral Surgery, Oral Medicine, and Oral Pathology will be published by the C. V. Mosby Company, of St. Louis, and will replace the Oral Surgery section of the American Journal of Orthodontics and Oral Surgery. Dr. Kurt Thoma, President of the American Academy of Oral Pathology, will be Editor-in-Chief and Editor for the New England Society of Oral Surgeons. Dr. Herman Becks will be Editor for the Seminar of Oral Medicine, and Dr. Hamilton Robinson will be Editor for the Academy. The Journal will appear in twelve issues per year.

The Academy of Oral Pathology will hold examination for fellowship in the Spring. Early announcement will be made of the exact time and place.

HAMILTON B. G. ROBINSON, *Editor*
Ohio State University
College of Dentistry
Columbus 10, Ohio

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